2. Cellular automata: the game of life and the SIRS model

In this Section we consider an important set of models used in computer simulations, which are called "cellular automata" (these are very similar to the so-called "agent based models"). A cellular automaton is normally defined on a discrete lattice (this is not necessarily the case for agent based models, which are often defined in continuum space). The state of a cellular automaton is specified by the state of all cells/agents in the lattice. The cells/agents then evolve from one time step to the next according to a set of rules, which determine the automaton. These rules can be deterministic, or stochastic.

While superficially a simulation following the evolution of a stochastic cellular automaton may look similar to a standard Monte-Carlo simulation, as both entail the setup of a Markov chain, i.e. a rule to go from one state to the next, there are important differences. Mainly, cellular automata do not rely on the existence of an energy function, like standard Monte-Carlo simulations do; this is because they describe systems which are not in thermal equilibrium, unlike Monte-Carlo methods (as done in Section 1), whose ultimate aim is to sample states in thermal equilibrium, weighted according to the Boltzmann weight.

We will first discuss the Game of Life, which is arguably the paradigm of a cellular automaton, and then go on to consider the SIRS model. These constitute the topic of Checkpoint 2.

1.1. The Game of Life

The Game of Life is a famous cellular automaton, which was introduced by Conway in the 70's. It is defined on a square lattice, and its state is determined by the states of all cells in the lattice. Each of the cell can be either "dead", or "alive".

The evolution of the Game of Life on a lattice is fully deterministic and is set by the following, very simple, rules:

- Any live cell with less than 2 live neighbours dies.
- Any live cell with 2 or 3 live neighbours lives on to the next step.
- Any live cell with more than 3 live neighbours dies.
- Any dead cell with exactly 3 live neighbours becomes alive.

Note that in Game of Life, unlike in the Ising model (and in the SIRS model), it is conventional to consider as neighbours all cells which have at least a point in contact with a given cell; i.e. there are 8 neighbours for each cell (north, south, east, west, and the four neighbours along the lattice diagonals).

The dynamics in the Game of Life is called fully *parallel* and deterministic. The meaning of deterministic here is simple: there is no stochasticity in the rules of the Game of Life. As for *parallel*, this means that all cells are evolved to the $n+1$-th time steps *simultaneously*, based on the knowledge of the system state at the $n-$th time step. In other words, in a parallel update we loop through the

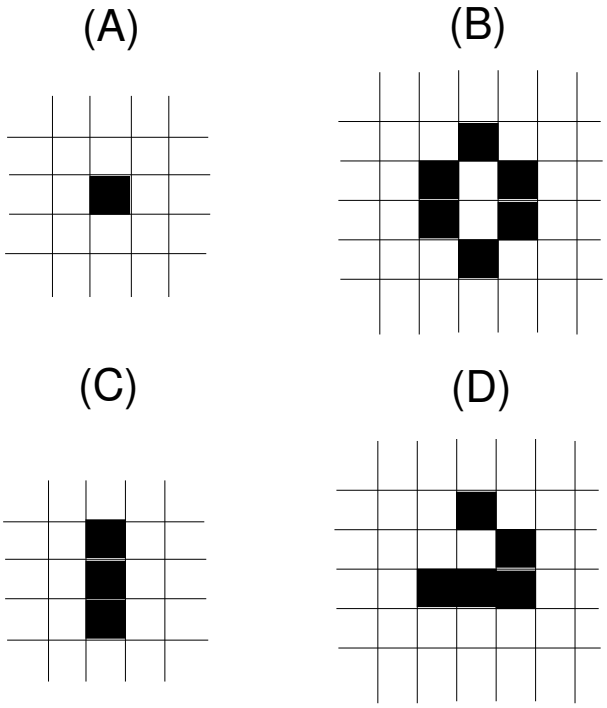

Figure 1: (A,B) Two situation leading to a static steady state (or absorbing state) in the Game of Life. The state in (A) will lead, in the next time step, to an empty lattice which can no longer evolve. The state in (B) remains unchanged, and is known as a "beehive". (C) Example of a state leading to an oscillatory steady state in the Game of Life. This is called a "blinker": in the next time step the blinker will transform into a row of 3 live cells, and in the following time steps it will evolve into the 3-cell column in (C) again. (D) A "glider": this is an example of a self-motile state, which, upon successive implementation of the rules of the Game of Life, moves (in this case along the diagonal of the lattice along the right and towards the bottom).

lattice, set the system state to a temporary variable, then update the new state to be this temporary variable. Another approach is to do a sequential update, which will be discussed in Section 1.2.

Now, given these dynamical rules, what is the emergent behaviour? Or, in other words, what is the behaviour of the system after many iterations, once *steady state* is achieved? This steady state is the analogue of the equilibrium state in the Monte-Carlo simulations discussed in Section 1.

There are three main possibility. First, the system may evolve into a state into which the dynamics gets stuck. This is an example of what is normally called, in nonequilibrium (i.e. non-thermal-equilibrium) statistical physics models, an *absorbing state.* Note that if the systems reaches an absorbing state, this means that the dynamics is not ergodic: if we were to start from the absorbing state, for instance, no other state can be reached by the dynamical update rules. An example of an absorbing state in the Game of Life is the empty lattice, in which all cells are dead – this is reached e.g. from the state in Fig. 1(A) at the next iteration. There are also quite non-trivial possible absorbing states, such as the "beehive" shown in Fig. 1(B).

A second possibility is that the system evolves into a perpetually oscillating steady state. Thus, the "blinker" in Fig. 1(C) is made up by a column of three neighbouring live cells. At the next update, the central cell stays alive, whereas the other two die as they only have 1 live neighbour each. However, the right and left neighbour of the central cell each have 3 live neighbours, hence they become alive the next time step, thereby maintaining the number of live cells equal to 3. The following time steps, a little thought will show that the state in Fig. 1(C) is reached again. As the system evolution is fully deterministic, the final outcome is that we will observe a never-ending oscillations between the 3-cell column and the 3-cell row. The period of oscillations of a blinker is 2 time steps. There exist many other more complicated oscillators, with longer periods.

Finally, an interesting state is the "glider" shown in Fig. 1(D). This more complicated pattern moves with constant velocity. A repeated application of the update rules shows that the glider here moves to the right and down, with configurations which cyclically repeat themselves. Within the Game of Life, self-motile patterns are called "spaceships".

The Game of Life originally came up as little more than a mathematician's game, but it has created the field of cellular automata. The most impressive patterns in the Game of Life are replicators, which reproduce themselves. These however are very complex indeed and are still being investigated to date!

1.2. The SIRS model

In the second Checkpoint we will consider another example of cellular automaton, the SIRS model, which is a model of epidemics spreading.

The SIRS comprises cells, or agents (i.e., people or other organisms), i, arranged on a two-dimensional lattice. The agents may contract some infection, and can be in one of three states:

- S: susceptible to the infection;
- I: infected;
- R: recovered from the infection (and hence immune to it).

The SIRS model is called like this because agents go through these states cyclically: S changes to I when a susceptible agent comes into contact with an infected one; recovery changes the agent from I to R; eventually, in the absence of contact with infected agents, the immune response wears off and R changes back to S.

We will consider in the Checkpoint a *stochastic*, rather than deterministic, version of this cellular automaton, at variance with what done when discussing the Game of Life in 1.1. The rules according to which the dynamics of the SIRS models unfolds are the following:

- a susceptible cell/agent S becomes infected (I) with probability p_1, if at least one nearest neighbour of i is I (SIRS uses the four nearest neighbours, as in the Ising model); otherwise the cell/agent remains susceptible.
- An infected site (I) becomes recovered (R) with probability p_2.
- A recovered site (R) becomes susceptible again with probability p_3.

Note that the dynamics becomes deterministic if $p_1 = p_2 = p_3 = 1$.

The typical update choice in the SIRS model is the so-called random sequential update. This means that to update the system we do as follows: (i) we select a cell randomly within the lattice (as when attempting to flip spins within the Ising model, when using the Glauber dynamics); (ii) we apply the SIRS rule to update the cell which we selected, looking at its current neaerst neighbours when deciding whether the cell should get infected. Therefore, just as in the Monte-Carlo simulation of the Ising model, it is natural to measure time in sweeps, where a sweep consists of N random sequential updates, with N the total number of cells in the lattice. Note that, as in the Ising model, because of the stochastic nature of the dynamics, during one single sweep one cell may be updated once, more than once, or never (but on average it will be updated once).

As in the case of the Game of Life, it is of interest to characterise the emerging behaviour of the system in steady state. Again, there are three possible scenarios.

First, the system can get stuck into an absorbing state. Whenever $p_3 \neq 0$, the only possible absorbing state is the one in which the whole lattice is made up by susceptible agent. Because each of these has no infected neighbours, there can be no further evolution starting from this state. (If $p_3 = 0$, any combination of susceptible and recovered agents is an absorbing state.)

Second, the system can evolve into a "dynamic equilibrium", where each of the agent cycles from S to I to R, then back to S. Overall, the average fraction of infected sites is non-zero in this regime.

Finally, close to the transition between absorbing phase and dynamic equilibrium, there are waves, where the infection spreads spatially to the whole lattice starting from a small infected region, then almost dies away, later on picks up again etc. These waves are also characterised by a non-zero fraction of infected sites, which, in this case, oscillates stochastically in time, and varies spatially. Waves arise due to the coupling between oscillations in time to spatial effects (spatial effects appear in the SIRS model due to the infection rules, which dictates that the infection spreads to nearest neighbours on the lattice).

All these states (absorbing phase, dynamic equilibrium, and waves) can only appear in nonequilibrium systems, and never in Markov chains set up to simulate systems in thermal equilibrium via Monte-Carlo (such as for the Ising model). Intuitively, this is because, once a system reaches thermal equilibrium, there is no way to say whether a movie showing successive configurations visited by the Markov chain is played forward or backward, as a system in thermodynamic equilibrium is invariant under time reversal. On the other hand, for all typical states of the SIRS model we can tell which way the movie goes. First, a system can dynamically enter an absorbing state, but never get out of it. Second, in the dynamic equilibrium we would expect to see agents cycle between susceptible to infected to recovered, but never from recovered to infected to susceptible (the causal relation would not work then!). Finally, waves have a well defined velocity and travel outwards during an outbreak, so that again one can tell the direction in which time progresses. In other words, the steady state in a nonequilibrium model such as the SIRS is *not* invariant under time reversal.

More rigorously, the reason why the steady states in nonequilibrium cellular automata are different to those observed in systems in thermal equilibrium is that the latter obey detailed balance, while the former do not. Recall that, for a system obeying detailed balance, the forward and backward transition probabilities from state μ to ν are linked to the steady state probabilities of observing each of the states, as follows (see Section 1 for a derivation and all definitions of symbols),

$$p_\mu P(\mu \to \nu) = p_\nu P(\nu \to \mu). \tag{1}$$

For example, a cell which cycles stochastically between states S, I, R and S with equal probability $p_1 = p_2 = p_3$ (this is similar to the dynamic equilibrium where we neglect spatial effects) clearly violates detailed balance: in steady state for instance $p_S = p_I = 1/3$ (indeed $p_S = p_I = p_R = 1/3$), while

$$\frac{P(I \to S)}{P(S \to I)} = 0 \neq \frac{p_S}{p_I} = 1. \tag{2}$$

Therefore the deep reason why nonequilibrium steady states like those observed for the SIRS model (and also for the Game of Life) are qualitatively different from those found in systems in thermal equilibrium is that away from equilibrium detailed balance no longer holds.

1.3. Characterising the SIRS model quantitatively

The SIRS model shows a phase transition between a state with no infection (the absorbing state, where all agents are susceptible in steady state), to another one where a finite fraction of the lattice is infected in steady state. This is an example of a nonequilibrium phase transition with an absorbing phase – as discussed previously no system in thermal equilibrium can ever display such a transition.

How can we discuss the phase transition in the SIRS model? From the study of the Ising model, we learnt that a useful way to characterise thermodynamic phase transition is via an order parameter. For the Ising model, this was the magnetisation per spin, which, in the thermodynamic limit ($N \to \infty$) is 0 in the disordered phase and different than 0 in the ordered phase. In the SIRS model, a natural choice is the fraction of infected sites, defined as follows

$$\psi = \frac{\sum_{j=1}^{N} \delta_{j,I}}{N}, \tag{3}$$

where $\delta_{j,I}$ is 1 if the $j-$th agent is infected, and 0 otherwise. In the Checkpoint, we will refer to the average fraction of infected sites as $\langle\psi\rangle$ or $\frac{\langle I\rangle}{N}$ – i.e., we will call $\langle I\rangle$ the average number of infected sites.

In the absorbing state, $\langle\psi\rangle = 0$, while $\langle\psi\rangle \neq 0$ when a dynamic equilibrium is achieved, or when waves appear. Here, the average $\langle\rangle$ can either be done by averaging over several uncorrelated measurements within the same simulation (as we did in the case of the Ising model), or by averaging over many different simulations. As for the Ising model, care needs to be taken to discard any transient (i.e. non-steady-state) behaviour. In the case of the SIRS model, after a system reaches the absorbing phase it never leaves it, therefore it makes sense to set the value of $\langle\psi\rangle = \frac{\langle I\rangle}{N} = 0$ for all cases where an absorbing state (without infected sites) is reached at any point in the simulation (this is because the system would stay there for an infinitely long time).

Fig. 2 shows some time series of the value of ψ for a single run, in the absorbing phase, in the case where dynamic equilibrium is achieved, and when waves arise. Each of these runs could then be averaged, as discussed above, to get a value of $\langle\psi\rangle$. In Checkpoint 2 we will build the phase diagram of the system, which consists in finding the value of $\langle\psi\rangle$ for generic values of p_1, p_2 and p_3 (for simplicity, we will fix the value of p_2). Within the phase diagram, a value of $\langle\psi\rangle = 0$ denotes the absorbing phase, where $\langle\psi\rangle \neq 0$ when the system reaches a dynamic equilibrium (or, as is sometimes said, is in the "active" phase). To pinpoint waves, we note that these are associated with large fluctuations of the value of ψ within a run (see Fig. 2(C)): these patterns can therefore be picked up by locating the peaks in the fluctuations of the order parameter, just as we did in Checkpoint 1 to locate the phase transition in the Ising model. Indeed, as previously mentioned, waves appear very close to the transition between the absorbing and the active phase. Note that, in analogy with the scaled specific heat, the properly normalised/scaled quantity to compute for fluctuations is the variance of the number of infected sites divided by N, or $\frac{\langle I^2\rangle - \langle I\rangle^2}{N}$ – as in the Ising model, this quantity is expected to be independent of N except at the

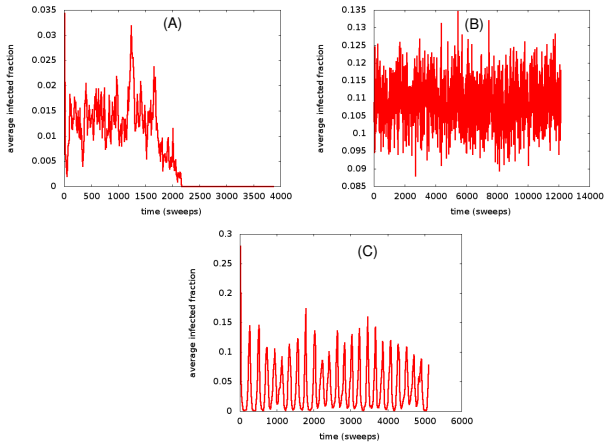

Figure 2: Example of the plot of $\psi = \frac{\langle I \rangle}{N}$ as a function of time (in units of sweeps) for a system evolving into the absorbing state (A); for a system reaching a dynamic equilibrium (B); and for a wave (C).

critical point.